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The Association Between Neurocognitive Functioning and Smoking in Adolescence: The TRAILS Study

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Objective: This study examines the association between neurocognitive functioning and tobacco smoking in adolescence. ***Method:*** Data from three measurements of the longitudinal Tracking Adolescents' Individual Lives Survey (TRAILS), a large regional population-based cohort study of Dutch adolescents, were used. The first measurement took place in 2001–2002 (T1) when participants were age 11, with two follow-up measurements (2003–2004 and 2005–2007; T2 and T3, respectively). A total of 1,797 adolescents participated in all three waves. At T1, they performed a selection of tasks from the Amsterdam Neuropsychological Tasks program (De Sonneville, 1999), which enabled the assessment of the main aspects of neurocognitive functioning. Smoking was assessed with a self-report questionnaire at T1, T2, and T3. In the multivariate analyses we controlled for gender, age, socioeconomic status (SES) and baseline speed. ***Results:*** Multivariate logistic regression analyses showed that poor sustained attention increased the likelihood that the adolescent would initiate smoking between T1 and T2. Low inhibition of prepotent responses increased the likelihood of smoking initiation between T1 and T3. An increased ability to inhibit biased response tendencies reduced the likelihood of being a daily smoker at T2. Poor sustained attention increased the likelihood of being a daily smoker at T3. ***Conclusion:*** Poor sustained attention and low inhibition predicted adolescent smoking. However, the proportion of the variance

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This research is part of the TRacking Adolescents' Individual Lives Survey (TRAILS). Participating centers of TRAILS include various departments of the University Medical Center and University of Groningen, the Erasmus University Medical Center Rotterdam, the University of Utrecht, the Radboud Medical Center Nijmegen, and the Parnassia Bavo group, all in the Netherlands. TRAILS has been financially supported by various grants from the Netherlands Organization for Scientific Research NWO (Medical Research Council program Grant GB-MW 940–38-011; ZonMW Brainpower Grant 100–001-004; ZonMw Risk Behavior and Dependence Grants 60–60600-98–018 and 60–60600-97–118; ZonMw Culture and Health Grant 261–98-710; Social Sciences Council medium-sized investment Grants GB-MaGW 480–01-006 and GB-MaGW 480–07-001; Social Sciences Council project Grants GB-MaGW 457–03-018, GB-MaGW 452–04-314, and GB-MaGW 452–06-004; NWO large-sized investment Grant 175.010.2003.005); the Sophia Foundation for Medical Research (projects 301 and 393), the Dutch Ministry of Justice (WODC), the European Science Foundation (EuroSTRESS project FP-006), and the participating universities. We are grateful to all adolescents, their parents, and teachers who participated in this research, and to everyone who worked on this project and made it possible. Zeena Harakeh was supported by a grant from the Netherlands Organization for Scientific Research (NWO) during the preparation of this article (Vernieuwingsimpuls, Veni: 451–08-018). Leo de Sonneville is director of Sonares BV, which produces the ANT software program used in this study.

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in smoking risk accounted for by these neurocognitive predictors proved to be small. Thus, although neurocognitive functioning is related to adolescent smoking, it seems to explain only a small part of why adolescents initiate and continue smoking.

Keywords: adolescent, tobacco, smoking onset, daily smoking, neurocognitive functioning

Adolescence is a period in which youth engage in risky behavior, such as smoking. It is evident that smoking has adverse health consequences, resulting in increased morbidity and mortality and costs to society (*www.who.org*). Therefore, it is important to identify potential precursors of smoking initiation and daily smoking in order to understand how the prevalence rate can be reduced. This will also provide insights for developing effective prevention programs (e.g., cognitive–behavioral interventions) to prevent and discourage adolescents from smoking. Although numerous studies have examined the role of various predictors including parent, peer, and school influences, as well as genetic influences (for reviews see Petraitis, Flay, & Miller, 1995; Tyas & Pederson, 1998), little is known about the role of neurocognitive factors in relation to smoking during adolescence.

Brain-imaging studies have shown that the (pre)frontal brain areas are developing and still maturing in the period from childhood to adolescence and even into early adulthood (Blakemore & Choudhury, 2006; Durston & Casey, 2006), until the age of 24 at least (Tau & Peterson, 2010). There are two important processes involved. First, during adolescence myelin (or white matter) layers continue to be formed around axons in the frontal and parietal cortices, and therefore, the transmission speed of neural information in these specific brain areas increases during this period (see Blakemore & Choudhury, 2006; Schepis, Adinoff, & Rao, 2008). Second, during adolescence, the synaptic density in the prefrontal cortex reaches a peak, followed by a period of synaptic pruning (i.e., connections that are frequently used are strengthened, while connections that are infrequently used are eliminated; see Blakemore & Choudhury, 2006; Schepis et al., 2008).

The changes in and maturation of parietal and prefrontal cortices during adolescence mean that the neurocognitive functions pertaining to self-regulation (e.g., attentional control, response switching, inhibition, and working memory), which rely on the functioning of these brain regions, are also still developing during adolescence (Blakemore & Choudhury, 2006). Besides the maturation of this cognitive-control system during adolescence, which is responsible for regulating impulses, the hormonal changes of puberty lead to an increased arousal of the socioemotional system (localized in limbic and paralimbic areas of the brain), which is sensitive to emotional and social stimuli and important for reward processing (Steinberg, 2007). The competition between these two systems makes adolescence a period in which risk taking is more common and constitutes a window of increased vulnerability to social and emotional stimuli from the environment, such as engagement in substance abuse like smoking (see Schepis et al., 2008; Steinberg, 2007). During adolescence there is an increase in the amount of time spent with peers; adolescents' risk-taking behavior such as smoking usually occurs in groups, as the presence of peers makes risk-taking behavior more rewarding (Steinberg, 2007). Therefore, adolescents with poor neurocognitive functioning may also have less self-regulation and control to resist tobacco smoking. They may therefore also be at increased risk for the effects of social and emotional stimuli (e.g., affiliation with deviant peers), which enhances engagement in smoking.

Most studies have examined substance use as a predictor of inabilities and impairments in neurocognitive functions but not vice versa. These findings show that, especially in adolescence, nicotine exposure from cigarette smoking is related to a deterioration in the prefrontal cortex activity, affecting neurocognitive functioning such as inhibition, attention, and verbal and working memory (Galván, Poldrack, Baker, McGlennen, & London, 2011; Lundqvist, 2005; Schepis et al., 2008). A relatively neglected but equally important perspective is whether neurocognitive functions may act as precursors for smoking during adolescence. Recently, a few longitudinal studies have focused on the relationship between neurocognitive factors, in particular regarding loss of self control or deficient self-regulation, and a combination of substance use (alcohol, tobacco, marijuana, and other drugs) or specifically alcohol or marijuana use.

These longitudinal studies focusing on substance use in general imply that neurocognitive functions may serve as a predictor for substance (ab)use and disorders. A longitudinal study by Tapert et al. (Tapert, Baratta, Abrantes, & Brown, 2002) was conducted among 66 high-risk youths who were approximately 15 years old at baseline. Their findings showed that *poor attention/neurocognitive functioning* in adolescents increased the chance of substance use (i.e., a combination of alcohol and other drug use) and dependence symptoms eight years later, even after controlling for covariates. Furthermore, the study of Tarter et al. (2003) compared neurobehavioral disinhibition for high-risk boys with a parent who met the American Psychiatric Association's *Diagnostic and Statistical Manual of Mental Disorders* (3rd ed., 1987; *DSM–III–R*) criteria for lifetime substance-use disorder and low-risk boys who did not have a parent with a lifetime substance-use disorder. The cross-sectional and longitudinal analyses among these 112 boys (10–12 years of age) showed that *neurobehavioral disinhibition* was prospectively associated with substance use (i.e., a combination of alcohol, tobacco, marijuana, and other drug use). Neurobehavioral disinhibition predicted the transition to substance-use disorder at age 19, but did not predict substance use at age 16. In a similar sample among 302 boys, the longitudinal study of Kirisci et al. (Kirisci, Tarter, Reynolds, & Vanyukov, 2006) showed similar findings. Neurobehavioral disinhibition was positively associated with engagement in substance use (i.e., a combination of alcohol, cannabis, and other drug use). Furthermore, higher neurobehavioral disinhibition was negatively related with the decision to desist from substance use. In line with these studies, disinhibition is shown to be a risk factor for alcohol use (Colder & O'Connor, 2002). In sum, these longitudinal findings show that poorer attention functioning and higher scores on neurobehavioral disinhibition are related to an increase in the development of substance (ab)use and disorder.

To the best of our knowledge there is only one longitudinal study that examined tobacco smoking specifically (Wilens et al., 2011). In contrast to the other studies that examined general substance use, the recent study of Wilens et al. (2011) showed that neurocognitive-function deficits did not predict stable smoking or substance-use disorders in older adolescents and young adults in an ADHD and non-ADHD sample. Moreover, among ADHD subjects, executive-function deficits did not pose any additional risk for substance-use disorders. The study of Wilens et al. (2011) used an overall measure of neurocognitive functioning. However, it would also be important to test each factor of neurocognitive functioning, separately and continuously, in order to provide more insight into subtle differences in neurocognitive functioning and smoking during adolescence. Furthermore, since previous studies were characterized by clinical and high-risk populations, little is known about neurocognitive functioning as a predictor of substance use such as smoking in population-based samples. In sum, little is known about the predictive association of neurocognitive functioning with regard to adolescent tobacco smoking. Moreover, nothing is yet known about this association regarding the early phases of smoking (i.e., smoking initiation).

It is important to focus on two different processes of smoking that adolescents may be engaged in: smoking initiation and daily smoking. Smoking initiation is more the experimentation phase of smoking; however, some adolescents become psychologically and physiologically dependent on tobacco and persist with smoking, which leads to daily smoking. Different predictors and underlying mechanisms may explain these two smoking processes, and thus the role of neurocognitive factors may differ between them.

The present study aims to provide knowledge of the association between neurocognitive functioning and tobacco smoking (i.e., smoking initiation and daily smoking) during a critical developmental period (ages 11–16 years). It is hypothesized that adolescents with poorer neurocognitive functioning (in particular those functions that support behavioral control) have a greater likelihood of engaging in tobacco smoking, that is, initiating smoking and persisting in daily smoking.

Method

Procedure and Participants

The present study is based on the data of the first, second, and third waves of the TRacking Adolescents' Individual Lives Survey (TRAILS). TRAILS was a prospective cohort study conducted among Dutch preadolescents at age 11. The participants were recruited from five municipalities in the north of the Netherlands, including both urban and rural areas. The sample selection involved two steps. First, the municipalities were requested to provide names and addresses of all inhabitants born between October 1, 1989 and September 30, 1990 (first two municipalities), or between October 1, 1990 and September 30, 1991 (last three municipalities), which yielded 3,483 names. Simultaneously, primary schools within these municipalities were approached with a request to participate. Of the 135 schools, 122 (90.4%) agreed to participate, accounting for 90.3% of the adolescents. More details about the procedure have been published elsewhere (De Winter et al., 2005). Of all the subjects who were approached ($N = 3145$), 6.7% were excluded because of severe mental or physical handicap or language problems. Of the remaining 2,935 young adolescents, 76.0% of the adolescents and their parents agreed to participate and were all enrolled in the study ($n = 2,230$, mean age 11.1 years, $SD = 0.6$, 50.8% girls [$N = 1,132$]). A high response rate (96%) was yielded at the second assessment after approximately two and a half years ($n = 2,149$, mean age 13.6 years, $SD = 0.5$, 51.2% girls [$N = 1,095$]). On the third assessment ($n = 1,816$, mean age 16.3 years, $SD = 0.7$, 52.3% girls [$N = 950$]), the response rate was somewhat lower (81.4% of the entire T1 sample). Attrition analyses showed that adolescents not participating at T3 did not significantly differ in smoking behavior ($p = .962$) or age ($p = .239$) from the ones who did participate at T3. However, significant differences were shown regarding gender ($p < .05$) and SES ($p < .001$). Girls and those with higher education levels were more likely to participate at T3 than were boys and those with lower education levels.

At each of the three assessments, the self-report questionnaires were completed by each adolescent. Most adolescents were seen in a group setting at their school or in designated testing locations. Some participants, who were unable to attend these measurement sessions, were visited at their homes. Confidentiality of the study was emphasized. In a workshop, undergraduate psychologists were trained by researchers from the study in how to administer the computerized tasks of the Amsterdam Neuropsychological Tasks (ANT) program (De Sonneville, 1999). The workshop was followed by practice administrations on several test cases, which were monitored. The observations and the quality of the collected practice data were evaluated to decide whether these trainees administered the tests correctly and according to the written protocol. Trainees who passed the quality-control phase were allowed to administer the tests and measurements in the TRAILS sample. Adolescents were tested individually for approximately 70 min (short breaks included) by these trained undergraduates in a separate room at their school (or, if this was not feasible, in a nearby community center). Quality control of collected data was conducted and information on the questionnaires was provided on a regular base throughout the testing phase.

Measures

Socioeconomic status (SES). SES was based on the parental report of income, educational, and occupational level of each parent at T1 (Ganzeboom & Treiman, 1996). After standardization, the mean of this scale was calculated. The Cronbach's alpha was 0.84. Subsequently, we categorized socioeconomic status into (a) low SES, representing the lowest quartile of scores (i.e., lowest score to −0.6589; reference group), (b) average SES, representing the middle two quartiles of scores (−0.6589 to 0.5719), and (c) high SES, representing the highest quartile of scores (0.5719 to the highest score; Amone-P'Olak et al., 2009; Veenstra, Lindenberg, Oldehinkel, de Winter & Ormel, 2006).

Neurocognitive functioning. The children performed five computerized tasks from the ANT (De Sonneville, 1999) to evaluate the main aspects of neurocognitive functioning (see Table 1 for a summary of the tasks used). These were (a) simple reaction time (RT), measured by baseline speed task; (b) automatic and controlled visuospatial pattern search (feature-identification task); (c) variability in task completion time, perceptual sensitivity and response bias (sustained-attention task); (d) working-memory ca-

Table 1
An Overview of the Five Subtests of the Amsterdam Neuropsychological Tasks

Task	Description (answering device: buttons of computer mouse)	Measure and description/operationalization
Baseline speed task	The simple reaction time/intensity (arousal) aspect of attention. The task consists of two parts one for left and one for right index finger, starting with the non-preferred index finger in the first part and the preferred index finger in the second part. On the computer screen, a cross is depicted that changes, at unexpected moments, into a square. When the participant sees the square s/he has to directly press the button of the mouse with the index finger. It takes 1 to 2 min for each part. Cognition is limited to the detection of the mere presence of the signal.	Baseline speed refers to the ability to detect and respond to a stimulus (simple reaction time). A higher score indicates a slower reaction time.
Feature-identification task	The recognition of abstract visuo-spatial patterns. The target pattern is a 3 × 3 matrix pattern which is ordered in a specific manner, with 3 red and 6 white compartments, and is shown during the instructions. Afterwards, four matrix patterns are depicted simultaneously each time, and the participant has to indicate whether the target pattern is among them by pressing the left (no) or right (yes) mouse button. The task takes approximately 4 to 6 min. Task conditions vary in degree of similarity between target pattern and distractors in the imperative signal. When similarity is high, controlled processing is required. When similarity is low, automatic processing suffices. See e.g. Huijbregts, De Sonneville, van Spronsen et al. (2002) for detailed task description.	Pattern search refers to the controlled, central cognitive ability to serially compare a particular visuo-spatial pattern with barely distinguishable (similar) visuo-spatial patterns, and was operationalized as the difference in reaction time on similar non-target and dissimilar non-target trials. A higher score indicates slow pattern search.
Sustained-attention task	The stimulus consists of a square displayed continuously at the center of the screen. At each trial, 3, 4 or 5 dots are displayed within the square. The target signal, requiring to press the "yes" key, contains 4 dots, the nontarget signal, requiring to press the "no" key, contains 3 or 5 dots. The participant is shown 600 signals, presented in 50 series of 12 trials. When the subject presses the wrong key, s(h)e hears a beep signal. Because the number of nontarget signals is twice the number of target signals (each series contains an equal number of 3, 4, and 5 dots), a response bias for the "no" key develops during time-on-task (see De Sonneville et al., 1994, for a detailed task description). The task takes approximately 14 to 20 minutes. Primary outcome parameters: completion time per series (50 values), number of errors per series. Sustained attention indices: variability in task completion time and in accuracy with time-on-task.	Variability in task completion time reflects the ability to maintain a stable performance over a prolonged period of time, and is operationalized as the within-subject *SD* of the 50 series' completion times. A higher variability in task-completion time indicates poor sustained attention. Perceptual sensitivity (time-on-task) refers to the perceptual sensitivity or ability to discriminate target signals from nontarget signals. Based on signal detection theory, d' was computed. Time-on-task perceptual sensitivity was operationalized as d' (first 120 trials)–d' (last 120 trials). A higher score indicates a decrease in perceptual sensitivity over time (poor sustained attention). Response bias (time-on-task) refers to the ability to inhibit (task-induced) biased response tendencies. Based on signal detection theory, β was computed. Response bias was operationalized as β (last 120 trials)–β (first 120 trials). A higher score indicates the ability to inhibit these biased response tendencies.
Memory-search task	The task consists of three parts. In each part, frames are presented with 4 letters. In the first part, participants have to indicate whether the frame contains a certain target letter by pressing the right/left mouse button (5–6 minutes). In the second (6–7 minutes) and third parts (8–9 minutes), memory load was increased to 2 and 3 letters respectively. Provides index for memory search capacity. See e.g. Huijbregts, Licht et al. (2002) for a detailed task description.	Working memory (WM) reflects the ability to maintain and compare increasing informational load in short-term memory. WM was evaluated by analysis of the performance change over Part1 to Part3. The scores were based on reaction time. Memory-search capacity was operationalized as mean RT (Part 3)–mean RT (Part 1). A higher score indicates a larger decrease in speed under high working memory demands, and thus poorer WM capacity.

Table 1 (*continued*)

Task	Description (answering device: buttons of computer mouse)	Measure and description/operationalization
Shifting-set task	A colored square jumps randomly on a horizontal bar to the right or left. Depending on the color of the square, the subject has to execute a compatible response (press the key in the direction of the jump) or an incompatible response (press the key in the opposite direction). This test consists of three parts. The first part requires only compatible responses (fixed compatible condition; 2–3 min), the second part requires only incompatible responses and it is imperative to inhibit the prepotent responses of the first part (fixed incompatible condition; 3–4 min). In the third part the color varies (random condition), requiring mental flexibility by continuously having to adjust the response set on the basis of the color of the square after the jump (6–8 minutes).	Inhibition of prepotent responses reflects the ability to inhibit an inappropriate, habitual response tendency. Inhibition was operationalized by: mean RT Part2 (in compatible trials)–mean RT part 1 (compatible trials). A higher score indicates poor (slow) inhibition of prepotent responses. Attentional flexibility refers to the ability to switch between two competing and unpredictable response sets. Attention flexibility was operationalized by: mean RT Part3 (compatible trials)–mean RT Part 1 (compatible trials). A higher score indicates slower switching and thus poorer attentional flexibility.

pacity (memory-search task); and (e) inhibition of prepotent responses and attentional flexibility (shifting-set task). The main outcome parameters were speed (RT) and accuracy of performance. Prior to analysis, RT and accuracy measures were converted into gender-specific and age-adjusted *z* scores (Brunnekreef et al., 2007). Table 1 gives an overview of the eight neurocognitive-function indices (description/operationalization) that were used in the analyses. Verbal task instructions were given before each task, emphasizing both speed and accuracy of performance. To ensure that the children understood these instructions, practice trials were performed preceding task assessment. The ANT has proven to be a sensitive and valid tool in nonreferred samples (e.g., Brunnekreef et al., 2007; Van der Heijden, Suurland, Swaab-Barneveld, & De Sonneville, 2011), as well as in referred samples of various clinical domains (e.g., Altink et al., 2008; Huijbregts, De Sonneville, Licht, Sergeant, & Van Spronsen, 2002; Huijbregts, De Sonneville, Van Spronsen, Licht, & Sergeant, 2002; Rowbotham, Pit-ten Cate, Sonuga-Barke, & Huijbregts, 2009; Van Rijn, Aleman, De Sonneville, & Swaab, 2009).

Smoking. Self-report measures of smoking were included at the three waves. When assessing smoking at (T1), adolescents were asked how often they had smoked cigarettes (during their lifetimes). Response categories were: 0 = *no/never*; 1 = *once*; 2 = *2–3 times*; 3 = *4–6 times*; 4 = *7 times or more*. When assessing smoking onset at both T2 and T3, adolescents were asked "Did you ever smoke, even if it was one cigarette or a few puffs (during your lifetime)?" Response categories were: 0 = *I have never smoked*; 1 = *I smoked once or twice*; 2 = *I smoke once in a while but not daily*; 3 = *I have smoked but quit*; and 4 = *I smoke daily*. Response categories were recoded into "never smoked" and "ever smoked" (including Response Categories 1 to 4). The latter category included the smokers, that is, adolescents who had smoked at least one or two times (e.g., Korhonen et al., 2010). Smoking onset was assessed by selecting the adolescents who never smoked a cigarette at T1 (the never smokers at T1) and examining whether they ever smoked at T2 or T3.

When assessing daily smoking at both T2 and T3, adolescents were asked "How many (rolled) cigarettes did you smoke on average in the previous month?" Responses were: 0 = *never smoked*; 1 = *did not smoke in the previous month*; 2 = *smoked less than one cigarette/week in the previous month*; 3 = *smoked less than one cigarette a day in the previous month*; 4 = *smoked 1–5 cigarettes a day in the previous month*; 5 = *smoked 6–10 cigarettes a day in the previous month*; 6 = *smoked 11–20 cigarettes a day in the previous month*; and 7 = *smoked > 20 cigarettes a day in the previous month*. Response categories were recoded into "not a daily smoker" (including response categories 0 to 3) and "daily smoker" (including Response Categories 4 to 7; e.g., Korhonen et al., 2010).

Data Analyses

We used the dataset that included participants who participated at all three measurement waves (N = 1,797). Outliers on the neurocognitive-function measures were removed from the dataset when (a) the *z* scores were greater than 4 or −4 (Stevens, 2002), and/or (b) performance results reflected more than 50% errors. There were outliers on each of the following neurocognitive measures: baseline speed (n = 29), pattern search (n = 27), variability in task completion time (n = 15), working memory (n = 12), inhibition of prepotent responses (n = 22), attentional flexibility (n = 58), perceptual sensitivity (n = 45), and response bias (n = 53). For the analyses, we included the adolescents who had no outliers on any of these neurocognitive measures; thus a total of 1,640 adolescents remained in the sample. The excluded participants did not differ from the remaining sample in gender or age (Brunnekreef et al., 2007).

Subsequently, two-tailed, logistic regression analyses were conducted in Stata (Version 7.0, College Station, TX) to examine whether neurocognitive functioning predicted adolescent smoking onset at age 13 (T2) and age 16 (T3). For these analyses, adolescents who never smoked a cigarette at T1 (the never smokers at T1) were selected and it was examined whether they had initiated smoking at T2 (approximately two and a half years later) or at T3 (approximately five years later). The analyses were conducted in two steps. The first step involved bivariate logistic regression analyses. In the second step, multivariate logistic regression analyses were performed, including only the significant predictors ($p < .05$) from the first step, while controlling for gender (0 = female, 1 = male), age, SES and baseline speed as possible covariates. Similar analyses (i.e., logistic regression analyses) were performed to examine whether neurocognitive functioning

predicted adolescent daily smoking at T2 and T3. Again, the same two steps of analyses were followed. However, when predicting daily smoking, the total group (smokers and nonsmokers) at T1 was included.

Furthermore, adolescents were nested within schools, and therefore share several characteristics, such as having the same teacher or being exposed to the same educational system. This may result in dependence between the observations, which, in turn, can influence standard errors, confidence intervals, and *p* values. In order to obtain correct 95% confidence intervals and *p* values in a clustered sample, robust standard errors were obtained using the Hubert/White sandwich estimate of variance as implemented in Stata 10.0. (For a similar approach, see also Monshouwer, Smit, De Graaf, Van Os, & Vollebergh, 2005.) That is, the intraclass correlations embedded in the data structure were accounted for in the parameter estimation (Skinner, Holt, & Smith, 1989).

Results

Descriptive Statistics

The prevalence numbers of smoking onset and daily smoking in our sample were as follows: Of the 1,416 children who did not smoke at T1, between T1 and T2, 70.6% ($n = 999$) did not initiate smoking, whereas 28.2% ($n = 399$) did initiate smoking (with 1.3%, or 18 missing values); between T1 and T3, 45.4% ($n = 643$) did not initiate smoking, whereas 47.3% ($n = 670$) did initiate smoking (with 7.3%, or103 missing values). Of the total group of children at T1 ($N = 1,640$): at T2, 92.4% ($n = 1,516$) were not daily smokers and 5.9% ($n = 97$) were daily smokers (with 1.7%, or 27 missing values); at T3, 71.3% ($n = 1,170$) were not daily smokers and 20.6% ($n = 338$) were daily smokers (with 8.1%, or 132) missing values).

The correlations between the neurocognitive measures are depicted in Table 2. For the RT measures (i.e., baseline speed, pattern search, variability in task-completion time, working memory, inhibition of prepotent responses, attentional flexibility), the correlations ranged from 0.10 to 0.45 (all p values < 0.001). The correlation for the two accuracy measures (i.e., perceptual sensitivity and response bias) in the two samples (i.e., never-smoked sample at T1, and the total sample), was 0.02 and 0.03 (*ns*). In sum, intercorrelations were generally weak, suggesting limited overlap between the measures.

Neurocognitive Functioning and Smoking Onset

Bivariate logistic regression analyses showed that, based on the neurocognitive-function measures (see Table 1 for an overview and description of these measures), variability in task-completion time during sustained attention predicted smoking initiation at approximately two and a half (T2) and five years (T3) after baseline (T1; see Table 3). The odds ratios (OR) were 1.21 and 1.18, respectively. Inhibition of prepotent responses and pattern search predicted smoking initiation only at T3. The ORs were 2.24 and 1.31, respectively. When testing these three significant predictors in a multivariate logistic regression analysis, controlling for gender, age, SES, and baseline speed (see Table 4), variability in task completion time remained a significant predictor of smoking onset at T2 only (OR = 1.22). More specifically, one unit increase in variability in task-completion time (i.e., poor sustained attention) increased the likelihood of smoking initiation by 1.2, compared with never smoking at T2. Inhibition of prepotent responses remained a significant predictor of smoking onset at T3 (OR = 2.13). One unit increase in inhibition of prepotent responses (i.e., poorer inhibition) increased the likelihood of smoking initiation at T3 by about twice.[1]

Neurocognitive Functioning and Daily Smoking

Bivariate logistic regression analyses showed that response bias predicted the likelihood of being a daily smoker at T2 only (OR = 0.86, see Table 3). Variability in task-completion time (i.e., poor sustained attention) predicted the likelihood of being a daily smoker at T3 only (OR = 1.32, see Table 3). When testing these two significant predictors in a multivariate logistic regression analysis, controlling for gender, age, SES, and baseline speed, response bias remained a predictor for being a daily smoker at T2 (OR = 0.84), and poor sustained attention remained a predictor for being a daily smoker at T3 (OR = 1.31; see Table 4). This indicates that an increased ability to inhibit biased response tendencies (i.e., a higher score on response bias) reduced adolescents' likelihood of being daily smokers two and a half years later. Poor sustained attention (i.e., larger variability in task-completion time) increased adolescents' likelihood of being daily smokers at T3 (approximately five years later).

Discussion

The present longitudinal study examined whether neurocognitive functioning is related to smoking onset and daily smoking among Dutch children, aged 11 years at baseline. The findings showed that when controlling for gender, age, SES, and baseline speed, poor sustained attention (variability in task completion time), and inhibition (inhibition of prepotent responses and response bias) predicted adolescent smoking. Poor sustained attention operated as a risk factor for both smoking onset and daily smoking. This means that a poor cognitive ability to maintain a stable performance over a prolonged period of time increased adolescents' likelihood of initiating smoking and persisting with smoking (i.e., becoming a daily smoker). These findings are in line with previous findings on marijuana use and general substance use (Tapert et al., 2002). A possible explanation may be that the adolescents with poor sustained attention also have deficient self-regulation and control, and have demonstrated poorer inhibition and less flexibility.

Although neither response bias (i.e., high scores indicating inhibition of biased response tendencies) nor poor inhibition of

[1] In addition, analyses were repeated with a slightly different operationalization for smoking onset, i.e., excluding the adolescents who had smoked only one or two cigarettes from the category "ever smoked." The results from these bivariate and multivariate logistic regression analyses showed that, of the neurocognitive measures, only variability in task completion time (i.e. poor sustained attention) predicted smoking onset. Poor sustained attention increased the likelihood of smoking initiation at approximately two and a half (T2) and five years (T3) after baseline (T1). Pattern search and inhibition of prepotent responses were no longer significant predictors.

Table 2
Correlations Between Neurocognitive Measures

	1	2	3	4	5	6	7	8
Reaction-time measures:								
1. Baseline speed		0.19***	0.29***	0.17***	0.11***	0.19***		
2. Pattern search	0.20***		0.38***	0.33***	0.23***	0.30***		
3. Variability in task-completion time	0.26***	0.37***		0.45***	0.24***	0.27***		
4. Working memory	0.16***	0.33***	0.45***		0.16***	0.20***		
5. Inhibition of prepotent responses	0.10***	0.24***	0.24***	0.16***		0.32***		
6. Attentional flexibility	0.18***	0.29***	0.25***	0.20***	0.32***			
Accuracy Measures:								
7. Perceptual sensitivity								0.02
8. Response bias							0.03	

Note. Below the diagonal, the correlations are depicted for the smoking-onset sample (n = 1,416), and above the diagonal for the daily smoking (total) sample (N = 1,640).
*** $p < .001$.

prepotent responses were consistently related to adolescent smoking onset or daily smoking over time, our analyses indicated that inhibition of biased response tendencies (sustained-attention task) is a protective factor, whereas poor inhibition of prepotent responses (set-shifting task) seemed to be a risk factor. These findings are, in part, in line with previous findings, showing that neurobehavioral disinhibition is related to an increase in the development of substance (ab)use and disorder, and alcohol use (Colder & O'Connor, 2002; Kirisci et al., 2006; Tarter et al., 2003). The findings of Tarter et al. showed that neurobehavioral disinhibition predicted transition to substance-use disorder at age 19, but not to substance use in mid-adolescence (age 16). Their finding suggests that neurobehavioral disinhibition is an indicator of liability to develop substance-use disorder (Tarter et al., 2003). However, disinhibition has been assessed differently in studies; some studies used neuropsychological tests, others used self-report questionnaires. In some studies, disinhibition was even viewed as extraversion, sensation seeking, or impulsivity (Colder & O'Connor, 2002; Kirisci et al., 2006). Therefore, it is difficult to make comparisons between findings of studies. The tasks used to test inhibition in our study were specifically set up to reduce measurement error and to assess subtle interindividual differences in information processing (Brunnekreef et al., 2007).

In our study, we distinguished between two phases of smoking in order to examine whether differential associations between neurocognitive functioning and different phases of tobacco use (onset and frequent use) exist. There is evidence suggesting that the predictors and underlying processes of the later phases of smoking (i.e., daily smoking and persistence) may differ from the early phases of smoking (i.e., smoking initiation and experimentation) (True et al., 1999). Our findings show that neurocognitive functioning predicts both phases of smoking; sustained attention affected smoking onset as well as daily smoking.

The longitudinal study of Wilens et al. (2011) focusing specifically on smoking, showed that neurocognitive function deficits did not predict stable smoking or substance-use disorders in late adolescents and young adults (including a non-ADHD sample and an ADHD sample). The Wilens et al. (2011) study differs in several aspects from our study: They specifically focused on substance-use disorders using the *Diagnostic and Statistical Manual of Mental Disorders* (4th ed., 1994; *DSM–IV*) criteria and identifying a small number of stable smokers (N = 38); moreover,

Table 3
Bivariate Logistic Regression Analyses of the Association Between Neurocognitive Functioning and Adolescent Smoking Onset and Daily Smoking

	Smoking onset[a]						Daily smoking					
	Time 2 (N = 1398)			Time 3 (N = 1313)			Time 2 (N = 1612)			Time 3 (N = 1507)		
	OR	95% CI	p	OR	95% CI	p	OR	95% CI	p	OR	95% CI	p
Baseline speed	1.77	0.09–34.08	0.707	0.45	0.04–5.22	0.527	0.19	0.00–38.96	0.538	4.62	0.26–83.44	0.300
Pattern search	1.29	0.96–1.73	0.096	1.31*	1.02–1.69	0.034	1.05	0.67–1.63	0.840	1.30	0.98–1.72	0.064
Variability in task completion time	1.21*	1.04–1.42	0.015	1.18*	1.02–1.38	0.029	1.21	0.96–1.53	0.108	1.32***	1.13–1.55	0.000
Perceptual sensitivity	1.15	0.94–1.41	0.174	1.15	0.94–1.40	0.166	0.84	0.60–1.16	0.289	0.95	0.76–1.19	0.655
Response bias	1.00	0.95–1.06	0.937	0.99	0.95–1.04	0.721	0.86**	0.77–0.95	0.003	1.03	0.99–1.07	0.204
Working memory	1.31	0.81–2.12	0.266	1.25	0.82–1.89	0.294	1.60	0.67–3.82	0.289	1.20	0.75–1.93	0.451
Inhibition of prepotent responses	1.82	0.96–3.45	0.066	2.24*	1.17–4.29	0.015	0.90	0.32–2.49	0.834	1.15	0.59–2.22	0.688
Attentional flexibility	1.06	0.65–1.72	0.819	1.26	0.84–1.91	0.267	1.70	0.65–4.43	0.280	1.16	0.73–1.84	0.543

Note. Analyses regarding smoking onset selected non-smokers at T1; analyses regarding daily smoking consists the total group.
[a] Smoking onset was operationalized as smoking at least one or two times.
* $p < .05$. ** $p < .01$. *** $p < .001$.

Table 4
Multivariate Logistic Regression Analyses of the Association Between Neurocognitive Functioning and Adolescent Smoking Onset and Daily Smoking

	Smoking onset[a]						Daily smoking					
	Time 2 (N = 1383)			Time 3 (N = 1299)			Time 2 (N = 1595)			Time 3 (N = 1490)		
	OR	95% CI	p	OR	95% CI	p	OR	95% CI	p	OR	95% CI	p
Age	1.22	0.99–1.51	0.068	1.24	0.99–1.54	0.058	1.36	0.89–2.07	0.156	1.14	0.89–1.45	0.299
Gender	0.73*	0.57–0.93	0.012	0.75*	0.60–0.94	0.012	0.66	0.41–1.07	0.095	0.71*	0.55–0.93	0.013
SES:												
Low SES (reference)	1.00			1.00			1.00			1.00		
Average SES	0.73*	0.56–0.94	0.016	0.76*	0.58–1.00	0.049	0.39***	0.25–0.63	0.000	0.65**	0.48–0.87	0.004
High SES	0.46***	0.34–0.62	0.000	0.62**	0.47–0.82	0.001	0.38**	0.22–0.66	0.001	0.40***	0.26–0.60	0.000
Baseline speed	0.49	0.02–13.29	0.675	0.11	0.01–1.93	0.132	0.12	0.00–20.57	0.424	0.45	0.02–11.67	0.633
Pattern search	—			1.13	0.84–1.52	0.431	—			—		
Variability in task completion time	1.22*	1.03–1.45	0.024	1.15	0.96–1.37	0.131	—			1.31**	1.10–1.55	0.002
Response bias	—			—			0.84**	0.75–0.93	0.001	—		
Inhibition of prepotent responses	—			2.13*	1.11–4.11	0.024	—			—		
R-square	2.3%			1.8%			4.8%			3.1%		

Note. Analyses regarding smoking onset selected nonsmokers at T1; analyses regarding daily smoking comprise the total group. Only the significant predictors from Table 3 were tested, while controlling for gender, age, SES and baseline speed.
[a] Smoking onset was operationalized as smoking at least one or two times.
* $p < .05$. ** $p < .01$. *** $p < .001$.

they included an older sample (predicting smoking in late adolescents and young adults) and a longer follow-up (4-year and 10-year follow-ups for males; 5-year and 11-year follow-ups for females). However, the most important difference compared with our study is that we measured several factors of neurocognitive functioning separately and in a continuous manner, and therefore we were able to gain more insights and information on the subtle differences of each of these factors separately. In our study we also found a number of neurocognitive factors that were not related to smoking: pattern search, perceptual sensitivity, working memory, and attentional flexibility. A possible explanation for why these neurocognitive factors did not predict adolescent smoking, is that smoking might be specifically associated with measures of behavioral control, such as poor sustained attention and inhibition. However, this is one of the first studies assessing the neurocognitive factors separately, and attempting to understand the underlying mechanisms and processes of neurocognitive functioning and adolescent smoking, and so future research is needed to replicate our findings.

Strength and Limitations

This study has three important strengths: That is, the large and unselected nature of our adolescent sample, the longitudinal approach, and the computerized tasks used, which enabled us to assess subtle interindividual differences in information processing. Besides the strengths, some limitations have to be mentioned. First, the accuracy measures showed poor test–retest reliabilities (i.e., intraclass correlations < 0.60), which may have added measurement error, leading to an underestimation of the associations between the accuracy measures of information processing and smoking. In contrast, the RT measures showed adequate to high test–retest reliabilities (i.e., intraclass correlations > 0.60; Brunnekreef, 2006). Second, it is possible that under- or overreporting will occur when adolescents are asked to report their own smoking behavior. This may be due to inaccurate recall or a reluctance to tell the truth because of social desirability (Hill, Boudreau, Amyot, Déry, & Godin, 1997; McKennel, 1980; Patrick et al., 1994). However, to obtain information on adolescent smoking, self-administered questionnaires are often used. This method has been shown to be as reliable and valid as a more objective method, such as biochemical verification of smoking (Dolcini, Adler, & Ginsberg, 1996; Hunter, Webber, & Berenson, 1980). Third, boys and those with lower education levels were more likely to drop out from this study, which meant a slightly higher prevalence rate of girls and those with higher education levels in the sample we analyzed. In addition, the sample consisted of a regional population-based cohort, retrieved from the north of the Netherlands. Therefore, it is necessary to be cautious in generalizing our findings to the entire Dutch population or to other populations. Finally, although the findings of this present longitudinal study show a predictive association between neurocognitive functioning and adolescent smoking, we cannot provide conclusions about the causality of this association. In addition, other third variables or moderators may be involved, which were not tested in this study. For example, adolescents with low attention or low inhibition may be more likely to have social or learning difficulties, and therefore have less affiliation with school, or greater difficulties in establishing and/or retaining good relationship ties with parents and peers. This may in turn lead to increased risk-taking behavior such as tobacco smoking.

Future Research

Although we found significant associations, it should be noted that the results, even for the sustained-attention task, explain only a small part of the variance of adolescent smoking onset and daily smoking in a regional population-based cohort (i.e., R^2 for sustained attention ranging from 0.3% to 0.9%). Thus, neurocognitive-functioning deficits were shown to be related to

adolescent smoking, but seem to explain only a small part of why adolescents take up and continue smoking. Certainly, in addition to neurocognitive functioning, environmental factors will play a part as well in explaining adolescent smoking. Moreover, interactions between neurocognitive functioning and environment may be important. Children with inabilities and impairments in neurocognitive functions may be more vulnerable to smoking-related influences in the social environment (such as peer, parental, or school factors). For example, adolescents with low self regulation and control, which are indices of poor sustained attention and inhibition, may give in more easily to engaging in substance use when they are reinforced by their peers. Future research is needed to understand whether adolescents with poorer neurocognitive functioning are perhaps more susceptible to smoking in certain social environments.

Besides the ANT, which serves as a sensitive and valid tool to measure aspects of information processing, it may also be important to examine decision-making processes regarding substance use. Motivation models assume that disinhibited individuals are mainly responding in anticipation or expectance of a reward (Colder & O'Connor, 2002), and this may provide an alternative or additional explanation of adolescent substance use that requires further study.

Conclusion

This study shows that poor sustained attention and low inhibition predicted adolescent smoking. This may have important clinical implications when developing and implementing antismoking interventions and programs. These programs should focus on those adolescents with attention and/or inhibition deficits and tailor antismoking messages and programs specifically for these adolescents by, for example, communicating short and interactive messages (Kirisci et al., 2006). However, though neurocognitive functioning is related to adolescent smoking, it seems to explain only a small part of the variance in smoking initiation and daily smoking.

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